AMENDMENTS TO THE SPECIFICATION

Please amend paragraph 0193 on pages 56-57 as follows:

[0001] The initial description of the BALB/cByJ-*fld* mutant mouse (hereafter known as *fld*) focused on two features for which the mutation was named—presence of a triglyceride-filled fatty liver and a progressive neuropathy affecting peripheral nerve (Langner *et al.* (1989) *J. Biol. Chem.* 264: 7994-8003; Langner *et al.* (1991) *J. Biol. Chem.* 266: 11955-11964; Rehnmark *et al.* (1998) *J. Lipid Res.* 39: 2209-2217; Klingenspor *et al.* (1999) *J. Biol. Chem.* 274: 23078-23084). Subsequently, an independent mutant strain, C3H/HeJ-*fld*^{2J} (known as *fld*^{2J}), with the same phenotype and a mutation allelic to *fld* was isolated (Mouse Genome Informatics Project: The Jackson Laboratory: Bar Harbor: Maine. (~~URL:http://www.informatics.jax.org~~ www.informatics.jax.org) (1999)). Our further characterization indicates that in addition to fatty liver and neuropathy, both *fld* and *fld*^{2J} mutants exhibit markedly diminished adipose tissue depots with 50-90% reductions in white and brown fat pad mass (Fig. 1*a* and data not shown). Adipocytes in the affected tissue appear immature, with sparse lipid droplets (compare Fig. 1*b* and *c*). Similar reductions in adipose tissue mass and cellular lipid content have been observed in transgenic mice expressing a dominant negative transcription factor that interferes with adipocyte differentiation (Shimomura *et al.* (1998) *Genes. Dev.* 12: 3182-3194). In these mice, it has been shown that reduction in leptin levels that results from diminished adipose tissue mass is responsible for the development of fatty liver, hypertriglyceridemia and insulin resistance (Shimomura *et al.* (1999) *Science* 401: 73-76). Analogously, *fld* mice exhibit significantly reduced plasma leptin levels (Fig. 1*d*) and insulin resistance, suggesting that the primary defect in *fld* and *fld*^{2J} mice is impaired adipose tissue development, with other phenotypic features (*i.e.*, fatty liver, hypertriglyceridemia, insulin resistance) occurring as secondary manifestations of the mutation.